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 NEWS 18  Aug 08  NTIS has been reloaded and enhanced
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 NEWS 21  Aug 19  The MEDLINE file segment of TOXCENTER has been reloaded
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L4   ANSWER 1 OF 26     MEDLINE
2002152357  Document Number: 21881723.    PubMed ID: 11884419.   Cutting edge:
     inhibitory functions of the killer cell lectin-like receptor G1 molecule
     during the activation of mouse NK cells. Robbins Scott H; Nguyen Khuong B;
     Takahashi Nobuaki; Mikayama Toshifumi; Biron Christine A; Brossay Laurent.
     (Department of Molecular Microbiology and Immunology and Graduate Program
     in Pathobiology, Division of Biology and Medicine, Brown University,
     Providence, RI 02912. Gemini Science, San Diego, CA 92121. ) JOURNAL OF
     IMMUNOLOGY, (2002 Mar 15) 168 (6) 2585-9.  Journal code: 2985117R. ISSN:
     0022-1767. Pub. country: United States. Language: English.
AB   The killer cell lectin-like receptor G1 (KLRG1) is the mouse homolog of
     the rat mast cell function-associated Ag and contains an immunoreceptor
     tyrosine-based inhibitory motif in its cytoplasmic domain. In this study
     we demonstrate that both pathogenic and nonpathogenic in vivo activation
     of NK cells induces the expression of KLRG1 on their cell surface. Upon
     infection with murine CMV, this induction peaks between days 5 and 7 with
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about 90% of the NK cells expressing KLRG1. On day 1.5 post-murine CMV infection of C57BL/6 mice, the main producers of IFN-gamma are the KLRG1-negative NK cells. This effect has been recapitulated in vitro as we show that engagement of KLRG1 on a transfected NK cell line inhibits both cytokine production and NK cell-mediated cytotoxicity. Taken together, these data illustrate the crucial role played by KLRG1 during the termination of mouse NK cell activation.

L4 ANSWER 2 OF 26 CAPLUS COPYRIGHT 2002 ACS
2002:662677 Interactions of the **mast cell function-associated antigen** with the type I Fc.vepsiln. receptor. Song, Jinming; Hagen, Guy; Smith, Steven M. L.; Roess, Deborah A.; Pecht, Israel; Barisas, B. George (Department of Physiology, Colorado State University, Fort Collins, CO, 80523, USA). Molecular Immunology, 38(16-18), 1315-1321 (English) 2002. CODEN: MOIMD5. ISSN: 0161-5890. Publisher: Elsevier Science Ltd..

AB Clustering the **mast cell function-assocd. antigen** (**MAFA**), a membrane glycoprotein expressed on 2H3 cells, by its specific monoclonal **antibody** G63 substantially inhibits secretion normally triggered by aggregating these cells' Type I Fc.vepsiln. receptor (Fc.vepsiln.RI). To explore possible **MAFA**-Fc.vepsiln.RI interactions giving rise to this inhibition, we have studied by time-resolved phosphorescence anisotropy the rotational behavior of both **MAFA** and Fc.vepsiln.RI as ligated by various reagents involved in Fc.vepsiln.RI-induced degranulation and **MAFA**-mediated inhibition thereof. From 4 to 37 .degree.C the rotational correlation times (mean.+-.S.D.) of Fc.vepsiln.RI-bound, erythrosin-conjugated IgE resemble those obsd. for **MAFA**-bound erythrosin-conjugated G63 Fab, 82.+-.17 .mu.s and 79.+-.31 .mu.s at 4 .degree.C, resp. Clustering the Fc.vepsiln.RI-IgE complex by antigen or by anti-IgE increases the phosphorescence anisotropy of G63 Fab and slows its rotational relaxation. Lateral diffusion of G63 Fab is also slowed by antigen clustering of the receptor. Taken together, these results suggest that unperturbed **MAFA** assocs. with clustered Fc.vepsiln.RI. They are also consistent with its interaction with the isolated receptor, a situation also suggested by FRET measurements on the system.

L4 ANSWER 3 OF 26 MEDLINE
2002460086 Document Number: 22207473. PubMed ID: 12217400. An unusual inhibitory receptor-the **mast cell function-associated antigen** (**MAFA**). Abramson Jakub; Xu Rong; Pecht Israel. (Department of Immunology, The Weizmann Institute of Science, 76100, Rehovot, Israel.) MOLECULAR IMMUNOLOGY, (2002 Sep) 38 (16-18) 1307. Journal code: 7905289. ISSN: 0161-5890. Pub. country: England: United Kingdom. Language: English.

AB The **mast cell function-associated antigen** (**MAFA**) is a type II membranal glycoprotein that was first identified on the surface of rat mucosal-type mast cells of the RBL-2H3 line. A C-type lectin domain and an immunoreceptor tyrosine-based inhibitory motif (ITIM) are located in the extracellular and intracellular domains of **MAFA**, respectively. Human and mouse homologues of **MAFA** have been discovered recently. However, they are expressed also or only by NK and T-cells, where they most probably play different roles. **MAFA** clustering by its specific **antibody** mAb G63 has been previously shown to cause a dose-dependent inhibition of the secretory response of these cells to the Fc varepsilon RI stimulus. More recent results established that **MAFA**'s inhibitory action involves at least two different enzymes: Following the tyrosyl-phosphorylation of **MAFA** ITIM by the PTK Lyn, two phosphatases SHIP and SHP2 are recruited to it at the plasma membrane where they propagate the inhibitory signals. The following is a brief report on this unusual inhibitory receptor and its functional activities.

L4 ANSWER 4 OF 26 CAPLUS COPYRIGHT 2002 ACS
2002:662674 An unusual inhibitory receptor-the **mast cell function-associated antigen** (**MAFA**).
Abramson, Jakub; Xu, Rong; Pecht, Israel (Department of Immunology, The Weizmann Institute of Science, Rehovot, 76100, Israel). Molecular Immunology, 38(16-18), 1307-1313 (English) 2002. CODEN: MOIMD5. ISSN: 0161-5890. Publisher: Elsevier Science Ltd..
AB The **mast cell function-assocd.** **antigen** (**MAFA**) is a type II membranal glycoprotein that was first identified on the surface of rat mucosal-type mast cells of the RBL-2H3 line. A C-type lectin domain and an immunoreceptor tyrosine-based inhibitory motif (ITIM) are located in the extracellular and intracellular domains of **MAFA**, resp. Human and mouse homologues of **MAFA** have been discovered recently. However, they are expressed also or only by NK and T-cells, where they most probably play different roles. **MAFA** clustering by its specific **antibody** mAb G63 has been previously shown to cause a dose-dependent inhibition of the secretory response of these cells to the Fc.vepsiln.RI stimulus. More recent results established that **MAFA**'s inhibitory action involves at least two different enzymes: Following the tyrosyl-phosphorylation of **MAFA** ITIM by the PTK Lyn, two phosphatases SHIP and SHP2 are recruited to it at the plasma membrane where they propagate the inhibitory signals. The following is a brief report on this unusual inhibitory receptor and its functional activities.

L4 ANSWER 5 OF 26 MEDLINE DUPLICATE 1
2002086114 Document Number: 21650140. PubMed ID: 11790110. The **mast cell function-associated** **antigen** and its interactions with the type I Fcepsilon receptor.
Song Jinming; Hagen Guy M; Roess Deborah A; Pecht Israel; Barisas B George. (Department of Chemistry, Colorado State University, Fort Collins, Colorado 80523, and Department of Immunology, The Weizmann Institute of Science, 76100 Rehovot, Israel.) BIOCHEMISTRY, (2002 Jan 22) 41 (3) 881-9. Journal code: 0370623. ISSN: 0006-2960. Pub. country: United States. Language: English.
AB Rat mucosal-type mast cells of the RBL-2H3 line express a glycoprotein termed the **MAst cell Function-** **associated Antigen** (**MAFA**). When **MAFA** is clustered by its specific monoclonal **antibody** G63, secretion normally triggered by aggregating these cells' type I Fcepsilon receptor (FcepsilonRI) is substantially inhibited. The nature of **MAFA** -FcepsilonRI interactions giving rise to this inhibition remains unclear. Rotational diffusion of a membrane protein is a sensitive probe of its involvement in intermolecular interactions. We have therefore studied by time-resolved phosphorescence anisotropy the rotational behavior of both **MAFA** and FcepsilonRI as ligated by various reagents involved in FcepsilonRI-induced degranulation and **MAFA**-mediated inhibition thereof. From 4 to 37 degrees C, the rotational correlation times (mean +/- SD) of FcepsilonRI-bound, erythrosin-conjugated IgE resemble those observed for **MAFA**-bound, erythrosin-conjugated G63 Fab, 82 +/- 17 and 79 +/- 31 micros at 4 degrees C, respectively. Clustering the FcepsilonRI-IgE complex by antigen or by anti-IgE increases the phosphorescence anisotropy of G63 Fab and slows its rotational relaxation. Lateral diffusion of G63 Fab is also slowed by antigen clustering of the receptor. Taken together, these results indicate that unperturbed **MAFA** associates with clustered FcepsilonRI. They are also consistent with its interaction with the isolated receptor.

L4 ANSWER 6 OF 26 MEDLINE DUPLICATE 2
2002327853 Document Number: 22066043. PubMed ID: 12069532. Mechanisms of protection induced by attenuated simian immunodeficiency virus. Stebbings Richard J; Almond Neil M; Stott E Jim; Berry Neil; Wade-Evans Alison M;

Hull Robin; Lines Jenny; Silvera Peter; Sangster Rebecca; Corcoran Terry; Rose Jane; Walker K Barry. (Division of Immunobiology, Division of Retrovirology, National Institute for Biological Standards and Control, Blanche Lane, South Mimms, Potters Bar, Hertfordshire, United Kingdom.. rstebbings@nibsc.ac.uk) . VIROLOGY, (2002 May 10) 296 (2) 338-53. Journal code: 0110674. ISSN: 0042-6822. Pub. country: United States. Language: English.

AB To determine whether attenuated simian immunodeficiency virus (SIV) vaccines confer protection against superinfection via secondary cellular immune responses, we searched for markers of immune activation following rechallenge. Productive infection with either attenuated SIVmacC8 or wild-type SIVmacJ5 resulted in a transient increase in T-lymphocyte CD25 and **Mafa**-DR expression. A pronounced increase in the frequency of FAS+ CD8+ lymphocytes was observed following SIVmacJ5 infection only. A transient increase in lymphocytes positive for intracellular IFN-gamma and IL-4 was observed following primary infection with either virus. In contrast, lymphocytes positive for intracellular IL-2 were reduced. Following SIVmacJ5 challenge of SIVmacC8-infected vaccinees, no evidence of detectable superinfection was obtained. Rechallenge of vaccinees did not alter the frequency of activated peripheral T-lymphocytes, perturb cytokine profiles, or generate an anamnestic **antibody** response. These data do not support the hypothesis that protection conferred by live attenuated SIV is mediated by the induction of vigorous T-cell responses upon rechallenge.

L4 ANSWER 7 OF 26 MEDLINE DUPLICATE 3

2002268791 Document Number: 22003813. PubMed ID: 12008030. Clustering the **mast cell function-associated antigen** (**MAFA**) leads to tyrosine phosphorylation of p62(Dok) and SHIP and affects RBL-2H3 cell cycle. Abramson Jakub; Pecht Israel. (Department of Immunology, The Weizmann Institute of Science, 76100, Rehovot, Israel.) IMMUNOLOGY LETTERS, (2002 Jun 3) 82 (1-2) 23-8. Journal code: 7910006. ISSN: 0165-2478. Pub. country: Netherlands. Language: English.

AB The **mast cell function-associated antigen** (**MAFA**) is a type II membranal glycoprotein expressed by rat mast cells and basophils. **MAFA** clustering by its specific monoclonal **antibody**, (mAb) G63, efficiently inhibits the FcvarepsilonRI induced secretory response of mucosal-type mast cells of the RBL-2H3 line, as well as bone marrow-derived mast cells. Here we present results which suggest that **MAFA** has also a capacity of modulating the cell cycle of the RBL-2H3 line. We found that **MAFA** clustering, by mAb G63 or by its F(ab')(2) fragments, reduces the cell proliferation rate. Cell cycle analysis by flow cytometry revealed that the number of cells in sub-G phase is considerably higher for cells on which **MAFA** was clustered. Results of biochemical experiments established that **MAFA** clustering leads to a marked increase in the transient tyrosine phosphorylation of the adaptor protein p62(Dok) and the inositol phosphatase SHIP. Concomitantly, their respective binding to RasGAP and Shc was increased. Furthermore, the GTP binding protein Sos1 was found to dissociate from Shc upon **MAFA** clustering, suggesting that SHIP and Sos1 compete for Shc binding. We therefore suggest that **MAFA** has also a role in regulating RBL-2H3 cell proliferation rate by inhibiting RasGTP formation in the Ras signaling pathway.

L4 ANSWER 8 OF 26 CAPLUS COPYRIGHT 2002 ACS

2001:713410 Document No. 135:271887 Soluble **mast cell function associated antigen** (**MAFA**) pharmaceutical compositions and methods of making and using them. Takahashi, Nobuaki; Mikayama, Toshifumi (Gemini Science, Inc., USA). PCT Int. Appl. WO 2001070805 A2 20010927, 49 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU,

CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US8596 20010316. PRIORITY: US 2000-PV190716 20000317.

AB This invention provides pharmaceutical compns. and methods for controlling and modifying Natural Killer (NK) cell and T cell functions by manipulation of "**mast cell function-assocd. antigen**", or "**MAFA**", polypeptide-mediated cell signaling. The invention provides pharmaceutical compns. and methods using an agent that specifically binds to an **MAFA** ligand on a target cell and prevents or inhibits NK- or T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell. Preventing or inhibiting an NK- or a T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell prevents or inhibits the cell surface **MAFA** from generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and prevents or inhibits the NK- or T cell-expressed cell surface **MAFA** from binding to a **MAFA** ligand or generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and inhibits an NK cell or T cell activity.

L4 ANSWER 9 OF 26 CAPLUS COPYRIGHT 2002 ACS

2000:191340 Document No. 132:217159 Control of the activity transmitted by a tyrosine kinase receptor. Daeron, Marc; Fridman, Wolf Herman; Malbec, Odile (Institut National De La Sante Et De La Recherche Medicale (I.N.S.E.R.M.), Fr.; Institut Curie). PCT Int. Appl. WO 2000016102 A1 20000323, 48 pp. DESIGNATED STATES: W: CA, JP, US; RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (French). CODEN: PIXXD2. APPLICATION: WO 1999-FR2165 19990910. PRIORITY: FR 1998-11372 19980911.

AB A means is disclosed for controlling the neg. regulation of an activation transmitted by a tyrosine kinase receptor (TKR), and in particular for controlling the neg. regulation of proliferation and/or differentiation of cells induced by a TKR. Such means comprise the use of compds. capable of co-aggregating a TKR with a receptor comprising at least an ITIM motif, and antagonist compds. of the co-aggregating compds.

L4 ANSWER 10 OF 26 CAPLUS COPYRIGHT 2002 ACS

2000:575044 Document No. 134:146030 Anti-major histocompatibility complex **antibody** responses in macaques via intradermal DNA immunizations. Dela Cruz, Charles S.; MacDonald, Kelly S.; Barber, Brian H. (Department of Immunology, University of Toronto, Toronto, ON, M5S 1A8, Can.). Vaccine, 18(27), 3152-3165 (English) 2000. CODEN: VACCDE. ISSN: 0264-410X. Publisher: Elsevier Science Ltd..

AB In simian immunodeficiency virus (SIV) models, immunization of macaques with uninfected human cells or human major histocompatibility complex (MHC) proteins can induce xenogeneic immune responses which can protect the animals from subsequent SIV challenges. These studies suggest that the induction of anti-MHC immune responses can be a viable vaccine strategy against human immunodeficiency virus type 1 (HIV-1). We have previously shown in mouse studies that DNA immunization with class I and class II MHC-encoding plasmids can elicit both xenogeneic and allogeneic **antibody** responses against conformationally intact MHC mols. (Vaccine 17 (1999) 2479-92). Here we take these observations one step closer to human applications and report that intradermal needle

immunizations of non-human primates with plasmid DNA encoding human MHC alleles can safely elicit xenogeneic anti-MHC **antibody** responses. Moreover, injecting macaques with DNA encoding a specific macaque allogeneic MHC induced anti-allogeneic MHC **antibodies** prodn. These studies show that DNA immunization with MHC-encoding vectors can indeed be used to induce specific anti-human xenogeneic, as well as anti-macaque allogeneic MHC immunity in non-human primates. This strategy could thus be used to mobilize anti-MHC **antibody** response which may be useful as part of an anti-HIV-1 vaccination approach.

L4 ANSWER 11 OF 26 MEDLINE DUPLICATE 4
2000203451 Document Number: 20203451. PubMed ID: 10741410. NK cell expression of the killer cell lectin-like receptor G1 (KLRG1), the mouse homolog of **MAFA**, is modulated by MHC class I molecules. Corral L; Hanke T; Vance R E; Cado D; Raulet D H. (Department of Molecular and Cell Biology and Cancer Research Laboratory, University of California, Berkeley 94720-3200, USA.) EUROPEAN JOURNAL OF IMMUNOLOGY, (2000 Mar) 30 (3) 920-30. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB Using a new mAb, 2F1, we characterize a mouse natural killer (NK) cell antigen termed 'killer cell lectin-like receptor G1' (KLRG1; formerly mouse **MAFA** or 2F1-Ag). KLRG1 is expressed on 30-60% of murine NK cells, and a small fraction of T cells, and is composed of a homodimer of glycosylated 30-38-kDa subunits. Strikingly, cell surface expression of KLRG1 by NK cells was substantially down-regulated in mice deficient for expression of class I molecules, in contrast to the Ly49 lectin-like NK receptors, which are up-regulated in class I-deficient mice. We could not demonstrate binding of KLRG1 to class I molecules in a cell-cell adhesion assay. Transgenic expression of KLRG1 under heterologous transcription elements was unaffected by class I deficiency, indicating that class I molecules do not affect the KLRG1 protein directly, and suggesting that regulation is at the level of expression of the endogenous KLRG1 gene. Evidence is presented that class I molecules regulate KLRG1 via interactions with class I-specific inhibitory Ly49 molecules and SHP-1 signaling. Thus, although KLRG1 and Ly49 molecules are both lectin-like inhibitory receptors that are regulated by class I expression, the effects of class I on the cell surface expression of the molecules are opposing, and the underlying regulatory mechanisms are distinct.

L4 ANSWER 12 OF 26 MEDLINE DUPLICATE 5
2000117678 Document Number: 20117678. PubMed ID: 10651806. An immunoreceptor tyrosine-based inhibitory motif, with serine at site Y-2, binds SH2-domain-containing phosphatases. Philosof-Oppenheimer R; Hampe C S; Schlessinger K; Fridkin M; Pecht I. (Departments of Organic Chemistry, The Weizmann Institute of Science, Rehovot, Israel.) EUROPEAN JOURNAL OF BIOCHEMISTRY, (2000 Feb) 267 (3) 703-11. Journal code: 0107600. ISSN: 0014-2956. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB Clustering of the **mast cell function-associated antigen** by its specific monoclonal **antibody** (G63) inhibits the FcepsilonRI-mediated secretory response. The cytosolic tail of the **mast cell function-associated antigen** contains a SIYSTL stretch, a potential immunoreceptor tyrosine-based inhibition motif. To investigate the possible functional role of this sequence, as well as identify potential intracellular proteins that interact with it, peptides corresponding to residues 4-12 of the **mast cell function-associated antigen**'s N-terminal cytoplasmic domain, containing the above motif, were synthesized and used in affinity chromatography of mast cell lysates. Both tyrosyl phosphorylated and thiophosphorylated **mast cell function-associated antigen** peptides bound the src homology domain 2 (SH2)-containing tyrosine phosphatases-1 (SHP-1), -2

(SHP-2) and inositol 5'-phosphatase (SHIP), though with different efficiencies. Neither the nonphosphorylated peptide nor its tyrosyl phosphorylated reversed sequence peptide bound any of these phosphatases. Point mutation analysis of **mast cell function -associated antigen** pITIM binding requirements demonstrated that for SHP-2 association the amino acid residue at position Y-2 is not restricted to the hydrophobic isoleucine or valine. Glycine and other amino acids with hydrophilic residues, such as serine and threonine, at this position also maintain this binding capacity, whereas alanine and acidic residues abolish it. In contrast, SHP-1 binding was maintained only when serine was substituted by valine, suggesting that the Y-2 position provides selectivity for peptide binding to SH2 domains of SHP-1 and SHP-2. These results were corroborated by surface plasmon resonance measurements of the interaction between tyrosyl phosphorylated **mast cell function-associated antigen** peptide and recombinant soluble SH2 domains of SHP-1, SHP-2 and SHIP, suggesting that the associations observed in the cell lysates may be direct. Taken together these results clearly indicate that the SIYSTL motif present in **mast cell function -associated antigen**'s cytosolic tail exhibits characteristic features of an immunoreceptor tyrosine-based inhibition motif, suggesting it is a new member of the growing diverse family of immunoreceptor tyrosine-based inhibition motif-containing receptors.

L4 ANSWER 13 OF 26 SCISEARCH COPYRIGHT 2002 ISI (R)

1999:521007 The Genuine Article (R) Number: 211RG. Characterization of the three immunoglobulin G subclasses of Macaques. Calvas P; Apoil P A; Fortenfant F; Roubinet F; Andris J; Capra D; Blancher A (Reprint). CHU PURPAN, IMMUNOL LAB, PAVILLON CHARLES LEFEBVRE, F-31059 TOULOUSE, FRANCE (Reprint); UNIV TOULOUSE 3, HOP PURPAN, IMMUNOGENET MOL LAB, F-31062 TOULOUSE, FRANCE; ETABLISSEMENT TRANSFUS SANGUINE PYRENEES GARONNE, LAB IMMUNOHEMATOL, TOULOUSE, FRANCE; UNIV TEXAS, SW MED CTR, MOL IMMUNOL CTR, DALLAS, TX; OKLAHOMA MED RES FDN, OKLAHOMA CITY, OK 73104. SCANDINAVIAN JOURNAL OF IMMUNOLOGY (JUN 1999) Vol. 49, No. 6, pp. 595-610. Publisher: BLACKWELL SCIENCE LTD. P O BOX 88, OSNEY MEAD, OXFORD OX2 0NE, OXON, ENGLAND. ISSN: 0300-9475. Pub. country: FRANCE; USA. Language: English.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Southern blot experiments with genomic DNA samples of rhesus monkeys and crab-eating macaques and human C gamma-specific probes indicated that the two macaque species studied here possessed three C gamma genes per haploid genome. By amplifying the cDNA from macaque-mouse hybridomas, the coding sequences of two different rhesus monkey immunoglobulin (IE)G subclasses, IgG1(rh) (C gamma 1(rh)) and IgG2(rh) (C gamma 2(rh)), and one crab-eating macaque IgG subclass IgG1(**mafa**) (C gamma 1(**mafa**)), were characterized. None of the 16 rhesus monkey-mouse hybridomas studied here secreted IgG of the third subclass IEG3(rh) (C gamma 3(rh)). The C gamma 3(rh) gene was partly characterized at the genomic level. The cDNA of the C gamma 3(rh) gene was amplified from mRNA of rhesus monkey peripheral blood mononuclear cells (PBMC). The results are analysed in terms of phylogenesis of the C gamma genes. The cDNA sequences coding for the C mu and the C kappa domains of rhesus monkey Ig were established and compared to their human and non-human primate counterparts.

L4 ANSWER 14 OF 26 SCISEARCH COPYRIGHT 2002 ISI (R)

1999:806990 The Genuine Article (R) Number: BN85P. The **mast cell function-associated antigen**, a new member of the ITIM family. Xu R (Reprint); Pecht I. WEIZMANN INST SCI, DEPT IMMUNOL, IL-76100 REHOVOT, ISRAEL (Reprint). CURRENT TOPICS IN MICROBIOLOGY AND IMMUNOLOGY (AUG 1999) Vol. 244, pp. 159-168. Publisher: SPRINGER-VERLAG BERLIN. HEIDELBERGER PLATZ 3, D-14197 BERLIN, GERMANY. ISSN: 0070-217X. Pub. country: ISRAEL. Language: English.

L4 ANSWER 15 OF 26 MEDLINE DUPLICATE 6
1999323287 Document Number: 99323287. PubMed ID: 10397158. Mast cell stimulation by co-clustering the type I Fc epsilon-receptors with **mast cell function-associated antigens**. Schweitzer-Stenner R; Engelke M; Licht A; Pecht I. (Institut fur Experimentelle Physik, Universitat Bremen, Germany.. stenner@theo.physik.uni-bremen.de) . IMMUNOLOGY LETTERS, (1999 May 3) 68 (1) 71-8. Journal code: 7910006. ISSN: 0165-2478. Pub. country: Netherlands. Language: English.

AB The secretory response of rat mucosal-type mast cells (line RBL 2H3) to stimuli produced by clustering or co-clustering two of its membranal components; the type I Fc epsilon receptor and the **mast cell function associated antigen** (**MAFA**) was investigated. The primary reagents employed for this purpose were Fab fragments of the monoclonal **antibodies** J17 and G63 specific to the above respective proteins. The Fabs were then aggregated by F(ab')2 fragments of mouse IgG specific goat **antibodies**. This reaction was assumed to yield predominantly three different bivalent clustering reagents. Namely, dimers of the Fc epsilon RI specific (J17-Fab)2; dimers of the **MAFA** specific, (G63-Fab)2 and bispecific (J17-Fab-G63-Fab) dimers. The observed cellular secretory response was analyzed by employing a model which accounts for the clustering and co-clustering of Fc epsilon RIs and **MAFAs** by the above protocols. Results of this analysis provided evidence that at least some of the **MAFA** molecules are physically associated with the Fc epsilon RI. As a consequence, clustering of **MAFA** and Fc epsilon RI by bispecific J17-Fab-G63-Fab dimers induces secretion at comparatively low concentrations of these reagents, though with a significantly lower maximal response than that caused by the respective monospecific reagent (J17-Fab)2. This result most likely reflects the inhibitory capacity of **MAFA**-Fc epsilon RI interaction.

L4 ANSWER 16 OF 26 CAPLUS COPYRIGHT 2002 ACS
1998:795041 Document No. 130:51333 Human **MAFA** peptides for treating inflammation and allergy. Hewitt, Ellen Louise; Lamers, Maria Bardina Antonia Cornelia; Lamont, Alan; Williams, David Hugh (Peptide Therapeutics Limited, UK). PCT Int. Appl. WO 9854209 A2 19981203, 44 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1998-GB1572 19980529. PRIORITY: GB 1997-11148 19970531.

AB This invention relates to polypeptides, nucleotide sequences, **antibodies** or fragments thereof, ligands and compns. and their use in the medical fields of inflammation and allergy, disease examples of which include rheumatoid arthritis and asthma. These human **MAFA** peptides prevent cell activation, i.e. interleukin 2 release from T cells and IgE-mediated degranulation of basophils, as well as prevention of tumor growth.

L4 ANSWER 17 OF 26 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 7
1998417506 EMBASE 2F1 antigen, the mouse homolog of the rat '**mast cell function-associated antigen**', is a lectin-like type II transmembrane receptor expressed by natural killer cells. Hanke T.; Corral L.; Vance R.E.; Raulet D.H.. D.H. Raulet, Department Molecular Cell Biology, Cancer Research Laboratory, University of California, Berkeley, Berkeley, CA 94720-3200, United States. raulet@uclink4.berkeley.edu. European Journal of Immunology 28/12 (4409-4417) 1998.
Refs: 37.

ISSN: 0014-2980. CODEN: EJIMAF. Pub. Country: Germany. Language: English. Summary Language: English.

AB Inhibitory lectin-like receptors expressed on the surface of hematopoietic cells are critically involved in regulation of their effector functions. Here we report that a novel mAb specific for mouse NK cells, 2F1, recognizes the mouse homolog of the **mast cell function-associated antigen** (**MAFA**), an inhibitory lectin-like transmembrane receptor expressed on rat mast cells. The 2F1 antigen (2F1-Ag) and rat **MAFA** are structurally highly conserved and contain a cytoplasmic motif similar to the immunoreceptor tyrosine-based inhibitory motif that is presumably utilized for inhibitory signaling. We also identified a human homolog that is closely related to the rodent **MAFA**/2F1-Ag proteins. Like rat **MAFA**, 2F1-Ag is probably encoded by a single gene, which exhibits relatively little polymorphism. Strikingly, while rat **MAFA** is considered a mast cell antigen, we have been unable to detect cell surface expression of 2F1-Ag by mouse mast cell lines, bone marrow-derived mast cells, or peritoneal mast cells. Furthermore, mouse bone marrow-derived mast cells were devoid of 2F1-Ag mRNA. Instead, we find that approximately 40% of mouse NK cells express 2F1-Ag. Thus, **MAFA**/2F1-Ag may modulate immunological responses on at least two different cell types bridging the specific and innate immune system.

L4 ANSWER 18 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

1998:107396 Document No.: PREV199800107396. The human-**MAFA** has alternatively spliced variants. Lamers, Maricke; Lamont, Alan; Williams, David. Peptide Ther., 321 Cambridge Sci. Park, Milton Road, Cambridge CB4 4WG UK. Immunology, (Dec., 1997) Vol. 92, No. SUPPL. 1, pp. 101. Meeting Info.: 5th Annual Congress of the British Society for Immunology Brighton, England, UK December 2-5, 1997 British Society for Immunology. ISSN: 0019-2805. Language: English.

L4 ANSWER 19 OF 26 MEDLINE DUPLICATE 8

97205358 Document Number: 97205358. PubMed ID: 9052862. Clustering the **mast cell function-associated antigen** (**MAFA**) induces tyrosyl phosphorylation of the Fc epsilonRI-beta subunit. Rong X; Pecht I. (Department of Immunology, The Weizmann Institute of Science, Rehovot, Israel.) IMMUNOLOGY LETTERS, (1996 Dec) 54 (2-3) 105-8. Journal code: 7910006. ISSN: 0165-2478. Pub. country: Netherlands. Language: English.

AB The **mast cell function associated antigen** (**MAFA**) is a membranal glycoprotein identified on the surface membranes of rat mucosal-type mast cells of the RBL-2H3 line by a monoclonal **antibody** (G63) binding to it. **MAFA** clustering by mAb G63 causes a dose-dependent inhibition of these mast cells' response to immunological stimulus provided by the type 1 Fc epsilon receptor (Fc epsilonRI) suppressing the biochemical processes coupling it to mediator secretion. The inhibition was found to take place upstream to the production of inositol phosphates and the transient increase in free cytosolic Ca2+ ion concentration, hence it probably interferes with the cascade at the level of the protein tyrosyl kinases (PTK) activity. We have therefore examined whether **MAFA** clustering affects protein tyrosyl phosphorylation of cell components and found that a time-dependent increase is caused in this modification of the Fc epsilonRI-beta chain. This constitutes the first evidence for the capacity of the clustered **MAFA** to enhance, on its own, biochemical changes in the mast cells, changes that are most probably related to its inhibitory signaling capacity. Moreover, that the observed phosphorylation changes are in the Fc epsilonRI-beta chain clearly indicates possible cross-talk between these two membrane components.

L4 ANSWER 20 OF 26 MEDLINE DUPLICATE 9

96152728 Document Number: 96152728. PubMed ID: 8566088. Proximity

relationships between the type I receptor for Fc epsilon (Fc epsilon RI) and the **mast cell function-associated antigen** (**MAFA**) studied by donor photobleaching fluorescence resonance energy transfer microscopy. Jurgens L; Arndt-Jovin D; Pecht I; Jovin T M. (Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.) EUROPEAN JOURNAL OF IMMUNOLOGY, (1996 Jan) 26 (1) 84-91. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB Clustering of the **mast cell function-associated antigen** (**MAFA**) on the surface of rat mucosal type mast cells line 2H3 (RBL-2H3) leads to suppression of the secretory response induced by the type I Fc epsilon receptor (Fc epsilon RI). In order to establish a possible association between **MAFA** and Fc epsilon RI we measured fluorescence resonance energy transfer (FRET) between the **MAFA**-specific monoclonal **antibody** (mAb) G63 and Fc epsilon RI-bound ligands as well as between Fc epsilon RI-bound ligands themselves using the donor photobleaching FRET (pbFRET) technique. Average FRET efficiencies between 6 and 9% were determined after low-temperature incubation with fluorescent dye conjugated mAb G63 bound to **MAFA** (donor) and IgE bound to Fc epsilon RI (acceptor) on RBL-2H3 cells. Subsequent cross-linking of IgE by a polyvalent antigen caused no change in FRET efficiencies. These results suggest that the **MAFA** is located in the vicinity of the Fc epsilon RI on resting cells, and that clustering of the Fc epsilon RI leads to no significant change in the proximity of the two molecular species. In view of the sequence motif identified in the cytosolic tail of the **MAFA** and the observed changes in its phosphorylation upon antigen stimulation (Guthmann et al., Proc. Natl. Acad. Sci. USA 1995, 92: 9397-9401), the present study suggests that the secretory response inhibition by **MAFA** interferes with the signal transduction cascade initiated via the Fc epsilon RI. An additional finding was that clustering of the Fc epsilon RI by antigen showed a clear increase in the efficiency of FRET between Fc epsilon RI-bound IgE molecules conjugated with fluorescent donor and acceptor.

L4 ANSWER 21 OF 26 CAPLUS COPYRIGHT 2002 ACS

1995:988111 Document No. 124:21788 cloning of human gene **mafa** to produce **mast cell function-associated antigen**, ligand analysis, and uses for inflammation and allergy prevention. Pecht, Israel; Guthmann, Marcelo D.; Tal, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9527734 A1 19951019, 52 pp. DESIGNATED STATES: W: HU, JP, US; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1995-US4258 19950406. PRIORITY: IL 1994-109257 19940408.

AB An isolated DNA sequence encoding a mammalian **mast cell function-assocd. antigen** (**MAFA**) is provided. A sol. deriv. of the **MAFA** is obtained by culturing a host cell transformed by a recombinant expression vector comprising a sequence of said DNA encoding a form of sol. **MAFA**, and may be used for screening potential ligands of the **MAFA**. The ligands, alone or in combination with the **MAFA**, may be used in pharmaceutical compns. for the prevention of inflammatory and allergic reactions.

L4 ANSWER 22 OF 26 MEDLINE DUPLICATE 10

96016176 Document Number: 96016176. PubMed ID: 7568140. A secretion inhibitory signal transduction molecule on mast cells is another C-type lectin. Guthmann M D; Tal M; Pecht I. (Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.) PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1995 Sep 26) 92 (20) 9397-401. Journal code: 7505876. ISSN: 0027-8424. Pub.

country: United States. Language: English.

AB Secretion of inflammatory mediators by rat mast cells (line RBL-2H3) was earlier shown to be inhibited upon clustering a membrane glycoprotein by monoclonal **antibody** G63. This glycoprotein, named **mast cell function-associated antigen** (**MAFA**), was also shown to interfere with the coupling cascade of the type 1 Fc epsilon receptor upstream to phospholipase C gamma 1 activation by protein-tyrosine kinases. Here we report that the **MAFA** is expressed as both a monomer and a homodimer. Expression cloning of its cDNA shows that it contains a single open reading frame, encoding a 188-amino acid-long type II integral membrane protein. The 114 C-terminal amino acids display sequence homology with the carbohydrate-binding domain of calcium-dependent animal lectins, many of which have immunological functions. The cytoplasmic tail of **MAFA** contains a YXXL (YSTL) motif, which is conserved among related C-type lectins and is an essential element in the immunoreceptor tyrosine-based activation motifs. Finally, changes in the **MAFA** tyrosyl- and seryl-phosphorylation levels are observed in response to monoclonal **antibody** G63 binding, antigenic stimulation, and a combination of both treatments.

L4 ANSWER 23 OF 26 MEDLINE DUPLICATE 11

95337856 Document Number: 95337856. PubMed ID: 7613222. A new member of the C-type lectin family is a modulator of the mast cell secretory response. Guthmann M D; Tal M; Pecht I. (Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.) INTERNATIONAL ARCHIVES OF ALLERGY AND IMMUNOLOGY, (1995 May-Jun) 107 (1-3) 82-6. Journal code: 9211652. ISSN: 1018-2438. Pub. country: Switzerland. Language: English.

AB A glycoprotein identified on RBL-2H3 cells as capable of inhibiting the secretory response induced by the type I Fc epsilon receptor was named **mast-cell-function-associated antigen** (**MAFA**). The amino acid sequence deduced from the cloned full-length cDNA has now shown that the **MAFA** has marked sequence homology with several members of the C-type (calcium-dependent) animal lectin family. The high conservation of cysteinyl residues suggests an important role for intrachain disulfide bonds in attaining its structure and biological activity. We further show that **MAFA** clustering by monoclonal **antibody** G63 also inhibits the de novo synthesis and secretion of interleukin-6 induced by the Fc epsilon RI stimulus. Though no ligand has yet been identified for the **MAFA**, experiments using antisense oligonucleotides suggest that this novel lectin may have a role in cell adhesion in addition to its immunomodulatory capacity.

L4 ANSWER 24 OF 26 MEDLINE DUPLICATE 12

95003758 Document Number: 95003758. PubMed ID: 7920029. **Antibodies** specific to membrane components of rat mast cells are cross-reacting with human basophils. Geller-Bernstein C; Berrebi A; Bassous Gedj L; Ortega E; Licht A; Pecht I. (Kaplan Hospital, Rehovot, Israel.) INTERNATIONAL ARCHIVES OF ALLERGY AND IMMUNOLOGY, (1994 Nov) 105 (3) 269-73. Journal code: 9211652. ISSN: 1018-2438. Pub. country: Switzerland. Language: English.

AB Murine monoclonal **antibodies** (mAbs) have previously been raised by us with specificity to the following plasma membrane components of rat mucosal mast cells (RBL-2H3 subline): (1) the alpha-subunit of the type I Fc epsilon receptor, (Fc epsilon RI); (2) a newly described membrane glycoprotein, distinct from any of the Fc epsilon RI subunits, yet affecting the cell's secretory response to Fc epsilon RI clustering and hence named mast cell functional antigen (**MAFA**), and (3) a glycolipid, GD1b present in the RBL-2H3 cell's plasma membrane. The cross-reactivity of these mAbs with human basophils (from both allergic and nonallergic children) was now examined by three different protocols:

(1) by microscopy (light and dark field) of double stained basophils by toluidine blue and by fluorescein-labeled anti-mouse **antibodies** as secondary ligands binding to the mAbs; (2) by flow cytometry (using directly labeled mAbs), and (3) by monitoring the binding of the 125I-radiolabeled mAbs. In order to exclude the possibility of the (intact) mAbs binding to the Fc gamma receptors, also present on human basophils, Fab and (Fab')2 fragments derived from the above respective mAbs were employed wherever necessary. The results show that the above described murine mAbs fragments do bind specifically to basophils obtained from allergic and nonallergic children. These **antibodies** may thus also be employed as tools for studying the human basophils function.

L4 ANSWER 25 OF 26 MEDLINE DUPLICATE 13

93353456 Document Number: 93353456. PubMed ID: 7688811. Antigenic determinants of human sperm tail fibrous sheath proteins. Jassim A; al-Zuhdi Y; Gray A. (Department of Immunology, London Hospital Medical College, UK.) JOURNAL OF REPRODUCTIVE IMMUNOLOGY, (1993 Apr) 23 (3) 281-95. Journal code: 8001906. ISSN: 0165-0378. Pub. country: Netherlands. Language: English.

AB Mouse anti-fibrous sheath antisera (**MAFA**) produced by immunizing mice with purified preparations of human sperm tail fibrous sheath (FS) reacted with the principal piece of less than 10% of freshly isolated spermatozoa which were immotile and probably dead. Following their demembranation by detergents or repeated freezing and thawing, all spermatozoa were stained. This was also demonstrated on spermatozoa dried onto slides, but the undiluted xenoantisera showed additional reactivity with the acrosomal zone (AZ). Using immunogold electron microscopy, the target antigens were ultrastructurally localized to the FS, and a few spermatozoa showed some reaction at the AZ subacrosomal perinuclear theca. Following titration of the **antibodies**, the anti-AZ-reaction became undetectable at a dilution of 1:20 while their reactivity with the principal piece continued to a 1:400-dilution. These results indicated that the xenoantisera probably contained an additional unrelated **antibody** component which reacted with the AZ. Western blotting and staining of purified FS with **MAFA** detected seven major protein bands with MW ranging between 25 kDa and 97.4 kDa. In human testes, the 1:50 diluted **MAFA** reacted with sperm tails only, indicating the late expression of the antigenic determinants during spermatogenesis. **MAFA** did not react with oesophagus, stomach, duodenum, ileum, nasal lining tissues, uterus, pericardium, pancreas, thyroid gland, or cultured fibroblasts. The xenoantisera did, however stain the skin epidermis and cultured keratinocytes which exhibited filamentous cytoplasmic staining although their target antigens could not be biochemically identified. These results indicate that the FS proteins express antigenic determinants which are not shared with other cytoskeletal elements within the sperm flagellum or a variety of somatic tissues.

L4 ANSWER 26 OF 26 MEDLINE DUPLICATE 14

91346112 Document Number: 91346112. PubMed ID: 1831652. Possible interactions between the Fc epsilon receptor and a novel **mast cell function-associated antigen**.
Ortega E; Schneider H; Pecht I. (Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.) INTERNATIONAL IMMUNOLOGY, (1991 Apr) 3 (4) 333-42. Journal code: 8916182. ISSN: 0953-8178. Pub. country: ENGLAND: United Kingdom. Language: English.

AB We have recently described a monoclonal **antibody**, mAb G63, which identifies a novel membrane component of mast cells. This antigen is a glycoprotein with an apparent molecular mass of 28-40 kd, and is present on the surface of rat mucosal and serosal mast cells. Its density on cells of the mucosal mast cell line RBL-2H3 is 1 - 2 x 10(4) copies per cell. Crosslinking of this membrane protein by the intact mAb G63 results in a pronounced inhibition of the Fc epsilon RI-mediated secretion of RBL-2H3

cells. Here we show that crosslinking this novel membrane component inhibits biochemical processes initiated by Fc epsilon RI aggregation, such as the hydrolysis of phosphatidylinositides, the influx of Ca2+ ions, and the synthesis and release of de novo formed inflammatory mediators. Furthermore, by fluorescence microscopy, we show that crosslinking of Fc epsilon RI-IgE complexes by multivalent antigen results in redistribution of the membrane component recognized by G63, leading to its co-localization with the aggregated Fc epsilon RI. This localization is inhibited by NaN3, but not by colchicine or cytochalasin D. Fc epsilon RI crosslinking also promotes internalization of this novel membrane component. Taken together these data suggest that the mast cell membrane component recognized by mAb G63 is involved in the Fc epsilon RI-mediated stimulation of these cells, and thus can be considered a **mast cell function-associated antigen** (**MAFA**).

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L8 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
2001:713410 Document No. 135:271887 Soluble mast cell function associated antigen (**MAFA**) pharmaceutical compositions and methods of making and using them. Takahashi, Nobuaki; Mikayama, Toshifumi (Gemini Science, Inc., USA). PCT Int. Appl. WO 2001070805 A2 20010927, 49 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US8596 20010316. PRIORITY: US 2000-PV190716 20000317.

AB This invention provides pharmaceutical compns. and methods for controlling and modifying Natural Killer (**NK**) **cell** and T cell functions by manipulation of "mast cell function-assocd. antigen", or "**MAFA**", polypeptide-mediated cell signaling. The invention provides pharmaceutical compns. and methods using an agent that specifically binds to an **MAFA** ligand on a target cell and prevents or inhibits NK- or T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell. Preventing or inhibiting an NK- or a T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell prevents or inhibits the cell surface **MAFA** from generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and prevents or inhibits the NK- or T cell-expressed cell surface **MAFA** from binding to a **MAFA** ligand or generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns.

and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and inhibits an **NK cell** or T cell activity.

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L11 ANSWER 1 OF 5 SCISEARCH COPYRIGHT 2002 ISI (R)
2002:492558 The Genuine Article (R) Number: 560AN. Clustering the mast cell function-associated antigen (**MAFA**) leads to tyrosine phosphorylation of p62(Dok) and SHIP and affects RBL-2H3 cell cycle. Abramson J; Pecht I (Reprint). Weizmann Inst Sci, Dept Immunol, IL-76100 Rehovot, Israel (Reprint). IMMUNOLOGY LETTERS (3 JUN 2002) Vol. 82, No. 1-2, Sp. iss. SI, pp. 23-28. Publisher: ELSEVIER SCIENCE BV. PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS. ISSN: 0165-2478. Pub. country: Israel. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The mast cell function-associated antigen (**MAFA**) is a type 11 membranal glycoprotein expressed by rat mast cells and basophils. **MAFA** clustering by its specific monoclonal antibody, (mAb) G63, efficiently inhibits the FcepsilonR1 induced secretory response of mucosal-type mast cells of the RBL-2H3 line, as well as bone marrow-derived mast cells. Here we present results which suggest that **MAFA** has also a capacity of modulating the cell cycle of the RBL-2H3 line. We found that **MAFA** clustering, by mAb G63 or by its F(ab')(2) fragments, reduces the cell proliferation rate. Cell cycle analysis by flow cytometry revealed that the number of cells in sub-G phase is considerably higher for cells on which **MAFA** was clustered. Results of biochemical experiments established that **MAFA** clustering leads to a marked increase in the transient tyrosine phosphorylation of the adaptor protein p62(Dok) and the inositol phosphatase SHIP. Concomitantly, their respective binding to RasGAP and She was increased. Furthermore, the GTP binding protein Sos1 was found to dissociate from Shc upon **MAFA** clustering, suggesting that SHIP and Sos1 compete for She binding. We therefore suggest that **MAFA** has also a role in regulating RBL-2H3 cell proliferation rate by inhibiting RasGTP formation in the Ras signaling pathway.

L11 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2002 ACS
2001:713410 Document No. 135:271887 Soluble mast cell function associated antigen (**MAFA**) pharmaceutical compositions and methods of making and using them. Takahashi, Nobuaki; Mikayama, Toshifumi (Gemini Science, Inc., USA). PCT Int. Appl. WO 2001070805 A2 20010927, 49 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US8596

20010316. PRIORITY: US 2000-PV190716 20000317.

AB This invention provides pharmaceutical compns. and methods for controlling and modifying Natural Killer (**NK**) **cell** and **T cell** functions by manipulation of "mast cell function-assocd. antigen", or "**MAFA**", polypeptide-mediated cell signaling. The invention provides pharmaceutical compns. and methods using an agent that specifically binds to an **MAFA** ligand on a target cell and prevents or inhibits NK- or **T cell**-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell. Preventing or inhibiting an NK- or a **T cell**-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell prevents or inhibits the cell surface **MAFA** from generating an inhibitory signal to the NK or the **T cell**. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a **T cell**-expressed cell surface **MAFA** and prevents or inhibits the NK- or **T cell** -expressed cell surface **MAFA** from binding to a **MAFA** ligand or generating an inhibitory signal to the NK or the **T cell**. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a **T cell**-expressed cell surface **MAFA** and inhibits an **NK cell** or **T cell** activity.

L11 ANSWER 3 OF 5 MEDLINE DUPLICATE 1

2001143205 Document Number: 21115136. PubMed ID: 11220622. Genomic structure, alternative splicing, and physical mapping of the killer cell lectin-like receptor G1 gene (KLRG1), the mouse homologue of **MAFA** . Voehringer D; Kaufmann M; Pircher H. (Institute for Medical Microbiology and Hygiene, Department of Immunology, University of Freiburg, Germany.) IMMUNOGENETICS, (2001) 52 (3-4) 206-11. Journal code: 0420404. ISSN: 0093-7711. Pub. country: United States. Language: English.

AB The mouse killer cell lectin-like receptor G1 (KLRG1), the mouse homologue of the mast cell function-associated antigen (**MAFA**), is an inhibitory C-type lectin expressed on natural killer (**NK**) **cells** and activated CD8 **T cells**. Here we report the complete nucleotide sequence, alternatively spliced variants, and the physical mapping of the KLRG1 gene in the mouse. The gene spans about 13 kb and consists of five exons. Short interspersed repeats of the B1 and B2 family, a LINE-1-like element, and a (CTT)170 triplet repeat were found in intron sequences. In contrast to human KLRG1 and to the murine KLR family members, mouse KLRG1 locates outside the NK complex on Chromosome 6 between the genes encoding CD9 and CD4.

L11 ANSWER 4 OF 5 MEDLINE DUPLICATE 2

2000203451 Document Number: 20203451. PubMed ID: 10741410. **NK cell** expression of the killer cell lectin-like receptor G1 (KLRG1), the mouse homolog of **MAFA**, is modulated by MHC class I molecules. Corral L; Hanke T; Vance R E; Cado D; Raulet D H. (Department of Molecular and Cell Biology and Cancer Research Laboratory, University of California, Berkeley 94720-3200, USA.) EUROPEAN JOURNAL OF IMMUNOLOGY, (2000 Mar) 30 (3) 920-30. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB Using a new mAb, 2F1, we characterize a mouse natural killer (**NK**) **cell** antigen termed 'killer cell lectin-like receptor G1' (KLRG1; formerly mouse **MAFA** or 2F1-Ag). KLRG1 is expressed on 30-60% of murine **NK cells**, and a small fraction of **T cells**, and is composed of a homodimer of glycosylated 30-38-kDa subunits. Strikingly, cell surface expression of KLRG1 by **NK cells** was substantially down-regulated in mice deficient for expression of class I molecules, in contrast to the Ly49 lectin-like NK receptors, which are up-regulated in class I-deficient mice. We could not demonstrate binding of KLRG1 to class I molecules in a

cell-cell adhesion assay. Transgenic expression of KLRG1 under heterologous transcription elements was unaffected by class I deficiency, indicating that class I molecules do not affect the KLRG1 protein directly, and suggesting that regulation is at the level of expression of the endogenous KLRG1 gene. Evidence is presented that class I molecules regulate KLRG1 via interactions with class I-specific inhibitory Ly49 molecules and SHP-1 signaling. Thus, although KLRG1 and Ly49 molecules are both lectin-like inhibitory receptors that are regulated by class I expression, the effects of class I on the cell surface expression of the molecules are opposing, and the underlying regulatory mechanisms are distinct.

L11 ANSWER 5 OF 5 MEDLINE DUPLICATE 3
1999077501 Document Number: 99077501. PubMed ID: 9862665. Virus-activated CD8 **T cells** and lymphokine-activated **NK cells** express the mast cell function-associated antigen, an inhibitory C-type lectin. Blaser C; Kaufmann M; Pircher H. (Institute for Medical Microbiology and Hygiene, Department of Immunology, University of Freiburg, Germany.) JOURNAL OF IMMUNOLOGY, (1998 Dec 15) 161 (12) 6451-4. Journal code: 2985117R. ISSN: 0022-1767. Pub. country: United States. Language: English.

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=> d his

     (FILE 'HOME' ENTERED AT 18:39:49 ON 10 SEP 2002)

     FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS' ENTERED AT 18:40:06 ON
     10 SEP 2002
L1          242 S MAST CELL FUNCTION ASSOCIATED ANTIGEN OR MAFA
L2           75 S L1 AND ANTIBODY
L3        57697 S L2 AND T CELL OR NK CELL
L4           26 DUP REMOVE L2 (49 DUPLICATES REMOVED)
L5            0 S L2 AND NK KILLING
L6           27 S MAFA AND NK CELL
L7            0 S L6 AND INHIBIT KILLING
L8            1 S L6 AND KILLING
L9           16 S L6 AND T CELL
L10           0 S L9 AND INHIBIT KILLING
L11           5 DUP REMOVE L9 (11 DUPLICATES REMOVED)

=> dup remove l6
PROCESSING COMPLETED FOR L6
L12           8 DUP REMOVE L6 (19 DUPLICATES REMOVED)

=> d l12 1-8 cbib abs

L12 ANSWER 1 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
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2002:492558 The Genuine Article (R) Number: 560AN. Clustering the mast cell function-associated antigen (**MAFA**) leads to tyrosine phosphorylation of p62(Dok) and SHIP and affects RBL-2H3 cell cycle. Abramson J; Pecht I (Reprint). Weizmann Inst Sci, Dept Immunol, IL-76100 Rehovot, Israel (Reprint). IMMUNOLOGY LETTERS (3 JUN 2002) Vol. 82, No. 1-2, Sp. iss. SI, pp. 23-28. Publisher: ELSEVIER SCIENCE BV. PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS. ISSN: 0165-2478. Pub. country: Israel. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

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L12 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2002 ACS

2001:713410 Document No. 135:271887 Soluble mast cell function associated antigen (**MAFA**) pharmaceutical compositions and methods of making and using them. Takahashi, Nobuaki; Mikayama, Toshifumi (Gemini Science, Inc., USA). PCT Int. Appl. WO 2001070805 A2 20010927, 49 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US8596 20010316. PRIORITY: US 2000-PV190716 20000317.

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cell-expressed cell surface **MAFA** and inhibits an **NK cell** or T cell activity.

L12 ANSWER 3 OF 8 MEDLINE DUPLICATE 1
2001143205 Document Number: 21115136. PubMed ID: 11220622. Genomic structure, alternative splicing, and physical mapping of the killer cell lectin-like receptor G1 gene (KLRG1), the mouse homologue of **MAFA** . Voehringer D; Kaufmann M; Pircher H. (Institute for Medical Microbiology and Hygiene, Department of Immunology, University of Freiburg, Germany.) IMMUNOGENETICS, (2001) 52 (3-4) 206-11. Journal code: 0420404. ISSN: 0093-7711. Pub. country: United States. Language: English.
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L12 ANSWER 4 OF 8 MEDLINE DUPLICATE 2
2000203451 Document Number: 20203451. PubMed ID: 10741410. **NK cell** expression of the killer cell lectin-like receptor G1 (KLRG1), the mouse homolog of **MAFA**, is modulated by MHC class I molecules. Corral L; Hanke T; Vance R E; Cado D; Raulet D H. (Department of Molecular and Cell Biology and Cancer Research Laboratory, University of California, Berkeley 94720-3200, USA.) EUROPEAN JOURNAL OF IMMUNOLOGY, (2000 Mar) 30 (3) 920-30. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.
AB Using a new mAb, 2F1, we characterize a mouse natural killer (**NK**) **cell** antigen termed 'killer cell lectin-like receptor G1' (KLRG1; formerly mouse **MAFA** or 2F1-Ag). KLRG1 is expressed on 30-60% of murine **NK cells**, and a small fraction of T cells, and is composed of a homodimer of glycosylated 30-38-kDa subunits. Strikingly, cell surface expression of KLRG1 by **NK cells** was substantially down-regulated in mice deficient for expression of class I molecules, in contrast to the Ly49 lectin-like NK receptors, which are up-regulated in class I-deficient mice. We could not demonstrate binding of KLRG1 to class I molecules in a cell-cell adhesion assay. Transgenic expression of KLRG1 under heterologous transcription elements was unaffected by class I deficiency, indicating that class I molecules do not affect the KLRG1 protein directly, and suggesting that regulation is at the level of expression of the endogenous KLRG1 gene. Evidence is presented that class I molecules regulate KLRG1 via interactions with class I-specific inhibitory Ly49 molecules and SHP-1 signaling. Thus, although KLRG1 and Ly49 molecules are both lectin-like inhibitory receptors that are regulated by class I expression, the effects of class I on the cell surface expression of the molecules are opposing, and the underlying regulatory mechanisms are distinct.

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L12 ANSWER 6 OF 8 MEDLINE DUPLICATE 4

1999077194 Document Number: 99077194. PubMed ID: 9862378. 2F1 antigen, the mouse homolog of the rat "mast cell function-associated antigen", is a lectin-like type II transmembrane receptor expressed by natural killer cells. Hanke T; Corral L; Vance R E; Raulet D H. (Department of Molecular and Cell Biology, University of California, Berkeley 94720-3200, USA.) EUROPEAN JOURNAL OF IMMUNOLOGY, (1998 Dec) 28 (12) 4409-17. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

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L12 ANSWER 7 OF 8 MEDLINE DUPLICATE 5

1999057052 Document Number: 99057052. PubMed ID: 9842918. **MAFA**-L, an ITIM-containing receptor encoded by the human **NK cell** gene complex and expressed by basophils and **NK cells**. Butcher S; Arney K L; Cook G P. (HGMP Resource Centre, Wellcome Genome Campus, Hinxton, GB.) EUROPEAN JOURNAL OF IMMUNOLOGY, (1998 Nov) 28 (11) 3755-62. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB The natural killer cell gene complex on human chromosome 12p12-13 encodes several C-type lectin receptor genes expressed by **NK cells** and other hematopoietic cells. We have identified a novel receptor gene in this region encoding a putative type II transmembrane glycoprotein. The product is 54% identical to the rat mast cell function-associated antigen (**MAFA**), which inhibits mast cell activation by IgE. The human **MAFA**-like receptor (**MAFA**-L) and the rat **MAFA** protein are expressed by basophils and both have an immunoreceptor tyrosine-based inhibitory motif in the cytoplasmic tail, consistent with an inhibitory role in basophil activation. Unlike rat **MAFA**, expression of the **MAFA**-L gene is not limited to mast cells and basophils. In common with other genes in the **NK**

cell gene complex **MAFA**-L is also expressed by natural killer cells as well as the monocyte-like cell-line U937. Expression in **NK cells** is restricted to peripheral blood **NK cells**, decidual **NK cells** do not express **MAFA**-L. While **MAFA**-L and rat **MAFA** might have a similar role in basophils, the expression of **MAFA**-L in other cell types implies additional functions for this molecule. The presence of the **MAFA**-L gene in the human **NK cell** complex indicates that this locus encodes C-type lectin receptors expressed by a variety of cells important in host defense.

L12 ANSWER 8 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
1999:219786 Document No.: PREV199900219786. Identification and characterization of the mouse "**MAFA**" homolog as an **NK cell** receptor. Hanke, Thomas (1); Corral, Laura (1); Raulet, David H. (1). (1) University of California, Berkeley, CA USA. Natural Immunity, (Feb., 1998) Vol. 16, No. 2-3, pp. 55. Meeting Info.: Fifth Annual Meeting of the Society for Natural Immunity Seventeenth International Natural Killer Cell Workshop Warrenton, Virginia, USA October 17-21, 1998 ISSN: 1018-8916. Language: English.

=> s MAFA and autoimmune
L13 1 MAFA AND AUTOIMMUNE

=> d l13 cbib abs

L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
2001:713410 Document No. 135:271887 Soluble mast cell function associated antigen (**MAFA**) pharmaceutical compositions and methods of making and using them. Takahashi, Nobuaki; Mikayama, Toshifumi (Gemini Science, Inc., USA). PCT Int. Appl. WO 2001070805 A2 20010927, 49 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US8596 20010316. PRIORITY: US 2000-PV190716 20000317.
AB This invention provides pharmaceutical compns. and methods for controlling and modifying Natural Killer (NK) cell and T cell functions by manipulation of "mast cell function-assocd. antigen", or "**MAFA**", polypeptide-mediated cell signaling. The invention provides pharmaceutical compns. and methods using an agent that specifically binds to an **MAFA** ligand on a target cell and prevents or inhibits NK- or T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell. Preventing or inhibiting an NK- or a T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell prevents or inhibits the cell surface **MAFA** from generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and prevents or inhibits the NK- or T cell-expressed cell surface **MAFA** from binding to a **MAFA** ligand or generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and inhibits an NK cell or T cell activity.

=> s MAFA and graft rejection

L14 0 MAFA AND GRAFT REJECTION

=> s MAFA and allogenic
L15 0 MAFA AND ALLOGENIC

=> s MAFA and suppression
L16 10 MAFA AND SUPPRESSION

=> dup remove 16
PROCESSING COMPLETED FOR L6
L17 8 DUP REMOVE L6 (19 DUPLICATES REMOVED)

=> d l17 1-8 cbib abs

L17 ANSWER 1 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
2002:492558 The Genuine Article (R) Number: 560AN. Clustering the mast cell function-associated antigen (**MAFA**) leads to tyrosine phosphorylation of p62(Dok) and SHIP and affects RBL-2H3 cell cycle. Abramson J; Pecht I (Reprint). Weizmann Inst Sci, Dept Immunol, IL-76100 Rehovot, Israel (Reprint). IMMUNOLOGY LETTERS (3 JUN 2002) Vol. 82, No. 1-2, Sp. iss. SI, pp. 23-28. Publisher: ELSEVIER SCIENCE BV. PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS. ISSN: 0165-2478. Pub. country: Israel. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
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L17 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2002 ACS
2001:713410 Document No. 135:271887 Soluble mast cell function associated antigen (**MAFA**) pharmaceutical compositions and methods of making and using them. Takahashi, Nobuaki; Mikayama, Toshifumi (Gemini Science, Inc., USA). PCT Int. Appl. WO 2001070805 A2 20010927, 49 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US8596 20010316. PRIORITY: US 2000-PV190716 20000317.
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L17 ANSWER 3 OF 8 MEDLINE DUPLICATE 1
2001143205 Document Number: 21115136. PubMed ID: 11220622. Genomic structure, alternative splicing, and physical mapping of the killer cell lectin-like receptor G1 gene (KLRG1), the mouse homologue of **MAFA** . Voehringer D; Kaufmann M; Pircher H. (Institute for Medical Microbiology and Hygiene, Department of Immunology, University of Freiburg, Germany.) IMMUNOGENETICS, (2001) 52 (3-4) 206-11. Journal code: 0420404. ISSN: 0093-7711. Pub. country: United States. Language: English.

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L17 ANSWER 4 OF 8 MEDLINE DUPLICATE 2
2000203451 Document Number: 20203451. PubMed ID: 10741410. **NK cell** expression of the killer cell lectin-like receptor G1 (KLRG1), the mouse homolog of **MAFA**, is modulated by MHC class I molecules. Corral L; Hanke T; Vance R E; Cado D; Raulet D H. (Department of Molecular and Cell Biology and Cancer Research Laboratory, University of California, Berkeley 94720-3200, USA.) EUROPEAN JOURNAL OF IMMUNOLOGY, (2000 Mar) 30 (3) 920-30. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

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L17 ANSWER 5 OF 8 MEDLINE DUPLICATE 3
1999077501 Document Number: 99077501. PubMed ID: 9862665. Virus-activated CD8 T cells and lymphokine-activated **NK cells** express the mast cell function-associated antigen, an inhibitory C-type lectin. Blaser C; Kaufmann M; Pircher H. (Institute for Medical Microbiology and Hygiene, Department of Immunology, University of Freiburg, Germany.) JOURNAL OF IMMUNOLOGY, (1998 Dec 15) 161 (12) 6451-4. Journal code: 2985117R. ISSN: 0022-1767. Pub. country: United States. Language: English.

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L17 ANSWER 6 OF 8 MEDLINE DUPLICATE 4
1999077194 Document Number: 99077194. PubMed ID: 9862378. 2F1 antigen, the mouse homolog of the rat "mast cell function-associated antigen", is a lectin-like type II transmembrane receptor expressed by natural killer cells. Hanke T; Corral L; Vance R E; Raulet D H. (Department of Molecular and Cell Biology, University of California, Berkeley 94720-3200, USA.) EUROPEAN JOURNAL OF IMMUNOLOGY, (1998 Dec) 28 (12) 4409-17. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB Inhibitory lectin-like receptors expressed on the surface of hematopoietic cells are critically involved in regulation of their effector functions. Here we report that a novel mAb specific for mouse **NK cells**, 2F1, recognizes the mouse homolog of the mast cell function-associated antigen (**MAFA**), an inhibitory lectin-like transmembrane receptor expressed on rat mast cells. The 2F1 antigen (2F1-Ag) and rat **MAFA** are structurally highly conserved and contain a cytoplasmic motif similar to the immunoreceptor tyrosine-based inhibitory motif that is presumably utilized for inhibitory signaling. We also identified a human homolog that is closely related to the rodent **MAFA**/2F1-Ag proteins. Like rat **MAFA**, 2F1-Ag is probably encoded by a single gene, which exhibits relatively little polymorphism. Strikingly, while rat **MAFA** is considered a mast cell antigen, we have been unable to detect cell surface expression of 2F1-Ag by mouse mast cell lines, bone marrow-derived mast cells, or peritoneal mast cells. Furthermore, mouse bone marrow-derived mast cells were devoid of 2F1-Ag mRNA. Instead, we find that approximately 40% of mouse **NK cells** express 2F1-Ag. Thus, **MAFA**/2F1-Ag may modulate immunological responses on at least two different cell types bridging the specific and innate immune system.

L17 ANSWER 7 OF 8 MEDLINE DUPLICATE 5
1999057052 Document Number: 99057052. PubMed ID: 9842918. **MAFA**-L, an ITIM-containing receptor encoded by the human **NK cell** gene complex and expressed by basophils and **NK cells**. Butcher S; Arney K L; Cook G P. (HGMP Resource Centre,

Wellcome Genome Campus, Hinxton, GB.) EUROPEAN JOURNAL OF IMMUNOLOGY, (1998 Nov) 28 (11) 3755-62. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB The natural killer cell gene complex on human chromosome 12p12-13 encodes several C-type lectin receptor genes expressed by **NK cells** and other hematopoietic cells. We have identified a novel receptor gene in this region encoding a putative type II transmembrane glycoprotein. The product is 54% identical to the rat mast cell function-associated antigen (**MAFA**), which inhibits mast cell activation by IgE. The human **MAFA**-like receptor (**MAFA**-L) and the rat **MAFA** protein are expressed by basophils and both have an immunoreceptor tyrosine-based inhibitory motif in the cytoplasmic tail, consistent with an inhibitory role in basophil activation. Unlike rat **MAFA**, expression of the **MAFA**-L gene is not limited to mast cells and basophils. In common with other genes in the **NK cell** gene complex **MAFA**-L is also expressed by natural killer cells as well as the monocyte-like cell-line U937. Expression in **NK cells** is restricted to peripheral blood **NK cells**, decidual **NK cells** do not express **MAFA**-L. While **MAFA**-L and rat **MAFA** might have a similar role in basophils, the expression of **MAFA**-L in other cell types implies additional functions for this molecule. The presence of the **MAFA**-L gene in the human **NK cell** complex indicates that this locus encodes C-type lectin receptors expressed by a variety of cells important in host defense.

L17 ANSWER 8 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
1999:219786 Document No.: PREV199900219786. Identification and characterization of the mouse "**MAFA**" homolog as an **NK cell** receptor. Hanke, Thomas (1); Corral, Laura (1); Raulet, David H. (1). (1) University of California, Berkeley, CA USA. Natural Immunity, (Feb., 1998) Vol. 16, No. 2-3, pp. 55. Meeting Info.: Fifth Annual Meeting of the Society for Natural Immunity Seventeenth International Natural Killer Cell Workshop Warrenton, Virginia, USA October 17-21, 1998 ISSN: 1018-8916. Language: English.

=> s (takahashi n?/au or mikayama t?/au)
L18 13876 (TAKAHASHI N?/AU OR MIKAYAMA T?/AU)

=> s l18 MAFA
MISSING OPERATOR L18 MAFA
The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s l18 and MAFA
L19 1 L18 AND MAFA

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L19 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
2001:713410 Document No. 135:271887 Soluble mast cell function associated antigen (**MAFA**) pharmaceutical compositions and methods of making and using them. **Takahashi, Nobuaki; Mikayama, Toshifumi** (Gemini Science, Inc., USA). PCT Int. Appl. WO 2001070805 A2 20010927, 49 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO

2001-US8596 20010316. PRIORITY: US 2000-PV190716 20000317.
AB This invention provides pharmaceutical compns. and methods for controlling and modifying Natural Killer (NK) cell and T cell functions by manipulation of "mast cell function-assocd. antigen", or "**MAFA**", polypeptide-mediated cell signaling. The invention provides pharmaceutical compns. and methods using an agent that specifically binds to an **MAFA** ligand on a target cell and prevents or inhibits NK- or T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell. Preventing or inhibiting an NK- or a T cell-expressed cell surface **MAFA** from binding to the **MAFA** ligand on the target cell prevents or inhibits the cell surface **MAFA** from generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and prevents or inhibits the NK- or T cell-expressed cell surface **MAFA** from binding to a **MAFA** ligand or generating an inhibitory signal to the NK or the T cell. The invention also provides pharmaceutical compns. and methods using an agent that specifically binds to an NK- or a T cell-expressed cell surface **MAFA** and inhibits an NK cell or T cell activity.

=> s l18 and "anti-MAFA"
L20 0 L18 AND "ANTI-MAFA"

=> s l18 and MAFA antibody
L21 0 L18 AND MAFA ANTIBODY

=> s l18 and antibody
L22 624 L18 AND ANTIBODY

=> s l22 and MAFA
L23 1 L22 AND MAFA

=> d l23 cbib abs

L23 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
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CA SUBSCRIBER PRICE	-8.67	-8.67

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1: Eur J Immunol 1998 Nov;28(11):3755-62

MAFA-L, an ITIM-containing receptor encoded by the human NK cell gene complex and expressed by basophils and NK cells.

Butcher S, Arney KL, Cook GP.

HGMP Resource Centre, Wellcome Genome Campus, Hinxton, GB.

The natural killer cell gene complex on human chromosome 12p12-13 encodes several C-type lectin receptor genes expressed by NK cells and other hematopoietic cells. We have identified a novel receptor gene in this region encoding a putative type II transmembrane glycoprotein. The product is 54% identical to the rat mast cell function-associated antigen (MAFA), which inhibits mast cell activation by IgE. The human MAFA-like receptor (MAFA-L) and the rat MAFA protein are expressed by basophils and both have an immunoreceptor tyrosine-based inhibitory motif in the cytoplasmic tail, consistent with an inhibitory role in basophil activation. Unlike rat MAFA, expression of the MAFA-L gene is not limited to mast cells and basophils. In common with other genes in the NK cell gene complex MAFA-L is also expressed by natural killer cells as well as the monocyte-like cell-line U937. Expression in NK cells is restricted to peripheral blood NK cells, decidual NK cells do not express MAFA-L. While MAFA-L and rat MAFA might have a similar role in basophils, the expression of MAFA-L in other cell types implies additional functions for this molecule. The presence of the MAFA-L gene in the human NK cell complex indicates that this locus encodes C-type lectin receptors expressed by a variety of cells important in host defense.

PMID: 9842918 [PubMed - indexed for MEDLINE]

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Eur J Immunol. 1996 Jan;26(1):84-91.

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Eur J Immunol. 1998 Dec;28(12):3755-3762

J Immunol 161: 6451-54, 1998

J Immunol 141(2): 4324-32; 1988

Natl Acad Sci USA 92: 9397-9401; 1995

Eur J Immunol 30: 920-930, 2000

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